Figure 21 is a table of 741 calcium channel antagonists according to the present invention.

DETAILED DESCRIPTION OF THE INVENTION

Biological systems in general are controlled by molecular interactions between bioactive ligands and their receptors, in which the receptor "recognizes" a molecule or a portion thereof (*i.e.*, a ligand domain) to produce a biological effect. The voltage-gated Ca^{++} channels are considered to be pharmacological receptors: they possess specific binding sites for ligands having agonist and antagonist activities; the binding of ligands to such sites allosterically modulates Ca^{++} flux through the channel; the channel properties (*i.e.*, gating and ion selectivity) are regulatable; and various channels are known to associate with G-proteins (D. Rampe and D.J. Triggle, *Prog. Drug Res.* 40: 191-238 (1993). Accordingly, diseases or conditions that involve, or are mediated by, Ca^{++} channels can be treated with pharmacologically active ligands that interact with such channels to initiate, modulate or abrogate transporter activity .

The interaction of a Ca^{++} channel and a Ca^{++} channel-binding ligand may be described in terms of "affinity" and "specificity". The "affinity" and "specificity" of any given ligand-Ca^{++} channel interaction is dependent upon the complementarity of molecular binding surfaces and the energetic costs of complexation (*i.e.*, the net difference in free energy ΔG between bound and free states). Affinity may be quantified by the equilibrium constant of complex formation, the ratio of on/off rate constants, and/or by the free energy of complex formation. Specificity relates to the difference in binding affinity of a ligand for different receptors.

The net free energy of interaction of such ligands with a Ca^{++} channel is the difference between energetic gains (enthalpy gained through molecular complementarity and entropy gained through the hydrophobic